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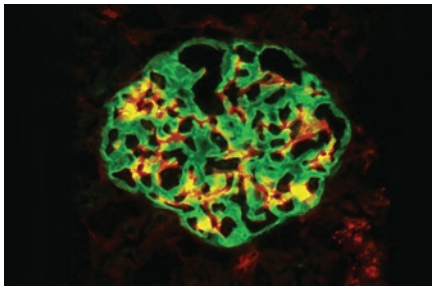
Heme oxygenase-1 protects podocytes in diabetes



The role of oxidant injury is now thought to be a common denominator in many, if not all, inflammatory and toxic diseases. Heme oxygenase-1 (HO-1) is an antioxidant enzyme normally upregulated in response to oxidant injury. As they report in this issue, Lee *et al.* induced diabetes in rats and found an increase in apoptosis of podocytes in their glomeruli. Accompanying or preceding these events was upregulation of HO-1 as well as several of the enzymes that cause apoptosis, such as caspase-3. The changes were blocked in cultured podocytes treated with high glucose by small interfering RNA for HO-1. *In vivo*, treatment of the diabetic rats with zinc protoporphyrin—an agent thought to act upstream of HO-1—reduced the number of apoptotic cells in the diabetic glomeruli. These results provide a direct test for the role of HO-1 in glomerular disease in diabetes. **See page 838.**

Licorice and hyperkalemia in ESRD

The plasma level of cortisol is an order of magnitude higher than that of aldosterone, but its affinity to the mineralocorticoid receptor is similar to that of aldosterone. Aldosterone is the actual mineralocorticoid due to the presence of the enzyme 11β-hydroxy-steroid dehydrogenase II, which converts cortisol into an inactive steroid and allows aldosterone to bind to its receptor in target tissues such as the collecting duct and colon. In a new study, Farese *et al.* treated 10 patients with ESRD with glycyrrhetinic acid derived from licorice, an inhibitor of the enzyme. This treatment allows cortisol as well as aldosterone to act on the major remaining aldosterone target tissue: the colon. Most patients demonstrated a reduction in serum potassium (K) before dialysis and in the frequency of severe hyperkalemia before dialysis. The study was conducted for 6 months; long-term studies are needed before the routine use of this treatment can be recommended. **See page 877.**

AKI predicts severity in MI

Acute myocardial infarction (MI) is often associated with acute kidney injury (AKI). To study its incidence and clinical significance, Goldberg *et al.* report on almost 2000 patients who survived an MI. A spectrum of elevated serum creatinine levels was found after MIs that occurred during hospitalization. Mild elevations, as expected, were transient. Creatinine elevations of more than 0.5 mg/dl above baseline were found in 138 patients and were transient in 60 of them. As compared with patients without AKI, the adjusted hazard ratio for mortality was 1.2 in patients with mild, transient AKI and 1.8 in patients with mild, persistent injury wherein creatinine levels remained elevated. Patients with persistent moderate/severe AKI had the highest mortality (hazard ratio 2.4). A similar relationship was present between AKI and admissions for heart failure. This study shows that the response of the kidney to acute MI is an excellent barometer of the health of the cardiovascular system. **See page 900.**